# **Epitomes**

# **Important Advances in Clinical Medicine**

# **Psychiatry**

The Scientific Board of the California Medical Association presents the following inventory of items of progress in psychiatry. Each item, in the judgment of a panel of knowledgeable physicians, has recently become reasonably firmly established, both as to scientific fact and important clinical significance. The items are presented in simple epitome and an authoritative reference, both to the item itself and to the subject as a whole, is generally given for those who may be unfamiliar with a particular item. The purpose is to assist busy practitioners, students, research workers or scholars to stay abreast of these items of progress in psychiatry that have recently achieved a substantial degree of authoritative acceptance, whether in their own field of special interest or another.

The items of progress listed below were selected by the Advisory Panel to the Section on Psychiatry of the California Medical Association and the summaries were prepared under its direction.

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### **Heterogeneity of Dementia**

THE CLINICAL SYNDROME of dementia is characterized by deficits in memory, judgment, language and other cognitive functions, as well as changes in personality and behavior. It is distinguished from delirium by the presence of clear consciousness in a patient. Dementia is etiologically heterogeneous, and a physician's first responsibility is to identify those patients whose dementia is caused by disorders amenable to specific treatments—for example, drug reaction, depression, metabolic dysfunction, infection, trauma, nutritional deficiency or neoplastic processes.

Unfortunately, the most frequent causes of dementia generally lead to progressively greater mental impairment despite symptomatic treatment. A study including 422 patients admitted to hospital with a diagnosis of dementia gave the following etiologic information: dementia of the Alzheimer type, 47 percent; multi-infarct type, 9 percent; alcohol related, 10 percent; normal-pressure hydrocephalus, 5 percent; depression, 4.5 percent; central nervous system tumors, 3.5 percent; hereditary chorea (Huntington's disease), 3.5 percent; drug toxicity, 2 percent; paralysis agitans (Parkinson's disease), Creutzfeldt-Jakob disease and other causes, each less than 1 percent.

Dementia of the Alzheimer type has begun to attract the most attention. It appears to increase in frequency with advancing age and is said to account for at least 20 percent of demented patients 80 years of age and older. A cholinergic deficit has become an accepted correlate of Alzheimer type dementia and yet in a significant number of clinically diagnosed patients involvement of the cholinergic system has not been found.

In contrast to the etiologic heterogeneity of the clinical syndrome of Alzheimer type dementia, one small autopsy series reported highly significant loss of neurons in the nucleus basalis (the major source of cholinergic brain innervation) in all cases, whereas a different group reported an equally striking loss of neurons in the locus ceruleus (the major source of noradrenergic brain innervation) in another small autopsy series.

The clinical diagnosis of Alzheimer type dementia remains largely one of exclusion: computerized tomographic scans are important to rule out other causes of dementia, electroencephalographic changes tend to be confirmatory rather than diagnostic and the clinical value of positron emission tomography (PET) and nuclear magnetic resonance (NMR) scans is yet to be established. The role of various etiologic agents (infectious, toxic metals, nutritional deficiencies and the like) remains questionable. Current treatment research focuses on correcting possible neurotransmitter imbalances and maximizing the function of surviving neurons. The search for etiologically distinct subgroups has been directed toward genetic markers (for example, haptoglobins and human leukocyte antigens), chromosomal abnormalities (for example, aneuploidy and sister chromatid exchange) and deficits in host defense mechanisms involving the immunoglobulin system and the philothermal response. Many treatments have been suggested for Alzheimer type dementia; so far, only those aimed at symptomatic relief have an acceptably high level of efficacy (for example, low doses of antipsychotic drugs for paranoid or agitated behavior, or both, and low doses of antidepressants for affective or vegetative concomitants of depression). Current experimental approaches include attempts to increase central nervous system concentrations of acetylcholine, chelating agents to lower aluminum concentrations, metabolic enhancers and treatments combining two or more approaches. None are particularly encouraging.

Patients with multiinfarct dementia may benefit from taking medications expected to reduce the incidence of further infarctions (for example, anticoagulants such as aspirin, 1½ grains every other day), though the prophylactic efficacy of this approach has not been proved. Of course, attempts should be made to control, at least in patients with multiinfarct dementia, risk factors for cerebrovascular disease, such as hypertension and smoking.

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### Phencyclidine (PCP) Psychosis

THE ABUSE of phencyclidine hydrochloride (known on the street as PCP, angel dust, wack, shermans or super kools) is common, perhaps epidemic, in most urban areas. Persons who have ingested large doses either through a single exposure or frequent use can have detectable concentrations of phencyclidine in their blood for months or even years afterward. The incidence of phencyclidine intoxication in patients presenting to urban psychiatric emergency services is alarming and usually underestimated. Commonly used laboratory detection methods (gas chromatographic techniques using flame ionization) are relatively insensitive with lowest limits of 50 to 100 ng per ml, whereas a glass capillary-gas chromatographic nitrogen detector method readily identifies phencyclidine in the 5 to 10 ng per ml range. Furthermore, phencyclidine screenings are commonly done on urine samples that are usually falsely negative if the urine is alkaline (pH above 6.0) even in the presence of high blood concentrations. Using the more sensitive techniques, phencyclidine has been detected in the blood of 40 percent to 70 percent of patients presenting to a Los Angeles public hospital psychiatric emergency service.

The psychiatric features of phencyclidine intoxication are protean and atypical. The commonly ascribed symptoms—toxic psychosis with agitation, disorientation, visual hallucinations and paranoid delusions coupled with nystagmus, tachycardia and hypertension—are found in less than 20 percent of phencyclidine-positive patients and are not related to blood concentrations of phencyclidine. Instead, patients have symptoms more typical of schizophrenia and affective disorder. Prospective comparisons of persons with laboratory tests positive for phencyclidine with those with negative results show no clinical or demographic features that

distinguish the two groups, except a history of phencyclidine use, which is obtained only 25 percent of the time. Optimum treatment of phencyclidine psychosis is not firmly established but involves chemotherapy appropriate to the behavioral and psychiatric symptoms and administration of ammonium chloride (0.5 grams three times a day) to acidify the urine and enhance excretion.

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## **Behavioral Treatment of Mild Hypertension**

WITH THE RECENT PUBLICATION of the findings of major clinical trials of antihypertensive drugs, it is now considered good practice to treat mild hypertension (diastolic pressure of 90 to 104 mm of mercury) as well as more severe cases. Given that 40 million people in the United States are estimated to have mildly elevated pressures, the economic and social consequences of such treatment are enormous. On close scrutiny of the design of the trials and the interpretation of the findings, however, serious questions have been raised about the wisdom of drug therapy in mild cases, especially for those persons free of cardiovascular risk factors such as smoking or hypercholesterolemia. Drug toxicity, disturbing side effects, possible adverse effects of longterm drug use and poor adherence are additional reasons for caution.

Nonpharmacologic modes of treatment are an alternative first step in uncomplicated cases. Weight loss and sodium restriction can be effective and are indicated in many cases, but poor adherence to diets no doubt reflects strong constitutional factors and the lifelong nature of eating habits.

Behavior patterns and psychologic and social stress have been shown to play a role in hypertension. Whether fundamental changes in behavior patterns and life-style can be achieved is uncertain. Nonetheless, people can be shown how to relax, trained to lower their blood pressure levels and advised on reducing responses to stress that facilitate blood pressure reduction.

Recent studies have shown that behavioral methods such as biofeedback and the regular practice of relaxation are effective, and studies of direct comparisons between drug and behavioral treatments have found comparable pressure reductions in many cases. Studies have shown that with the use of relaxation tapes and blood pressure biofeedback methods adapted for selfuse, behavioral methods can also be used effectively by persons at home, thereby facilitating regular practice and self-help in pressure reduction. Using small groups, hypertensive patients have also been taught how to manage stress more effectively. Finally, the simple pro-